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Ventricular morphology does not impact mid-term outcome after extracardiac total cavopulmonary connection

D. Laux*, M. Vergnat, R. Roussin, V. Lambert, M. Ly, M. Gouton, E. Belli

Centre des Malformations Congénitales Complexes-M3C-CCML, Centre Chirurgical Marie-Lannelongue, Département des cardiopathies congénitales, Le Plessis-Robinson, France

* Corresponding author.

E-mail address: d.laux@ccml.fr (D. Laux)

Background and objective Right ventricular morphology is a known risk factor for death in patients with univentricular hearts (UVH), especially before partial cavopulmonary connection. This study investigates the influence of anatomic and procedure related factors on mid-term outcome after extracardiac total cavopulmonary connection (TCPC).

Methods Retrospective single-center analysis of all patients with UVH undergoing extracardiac TCPC from 2000 to 2005. Patient and procedure related variables were analyzed.

Results 100 patients underwent extracardiac TCPC in the study period. 69% had a dominant left ventricle, 23% had a dominant right ventricle and 8% had biventricular anatomy. Median age at intervention was 6.1 years (1.1–29.4). At the moment of surgery, 9% were in NYHA class > 2 or had preoperative univentricular dysfunction. 6% had significant atrioventricular valve (AVV) regurgitation. Median preoperative mean pulmonary pressure was 12 mmHg (6–21). Early mortality was 4% and global mortality was 8%. Anatomic, functional, perioperative and postoperative parameters did not impact mortality. Only preoperative NYHA class > 2 was significantly associated to higher global mortality in univariate analysis. Freedom from death/transplantation was 92.6% (95% CI ± 0.05) at 5 years and 91.4% (95% CI ± 0.06) at 10 years after TCPC. Ventricular morphology did not influence global mortality (log rank $P=0.22$) or mid-term morbidity (thrombosis, protein losing enteropathy, arrhythmia; log rank $P=0.76$). Median follow-up was 8.9 years (0.01–14). At last visit, 96% of patients were in NYHA 1–2 with mostly (71%) normal univentricular function.

Conclusion Mid-term outcome after extracardiac TCPC is excellent with a good functional result and appears not to be influenced by ventricular morphology. Further studies are needed to evaluate the possible impact of ventricular morphology on late outcome after TCPC.

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Make use of time, let not advantage slip – How this William Shakespeare (1564–1616) quote lead us to optimize occlusion time for balloon dilatations –

X. Alacoque^{1,*}, R. Fesseau¹, K. Hadeed², G. Chausseray¹, S. Hascoet³, B. Leobon², P. Acar²

¹ CHU de Toulouse, Hôpital des enfants, Pédiatrie et Chirurgie viscérale, Toulouse, France



³ CHU Toulouse, Hôpital Purpan, Cardiologie, Toulouse, France

* Corresponding author.

E-mail address: alacoque.x@chu-toulouse.fr (X. Alacoque)

Introduction Neuro-developmental impairment which follows management of complex congenital heart disease is a major issue. Balloon dilation is a frequent procedure, which can lead to brain injury. Whereas the pressure and diameter of balloon for inflation are quite normalized, there are no written guidelines for time of inflation. Our aim was to study the brain perfusion with the NIRS (Near Infra Red Spectroscopy) during catheter procedure.

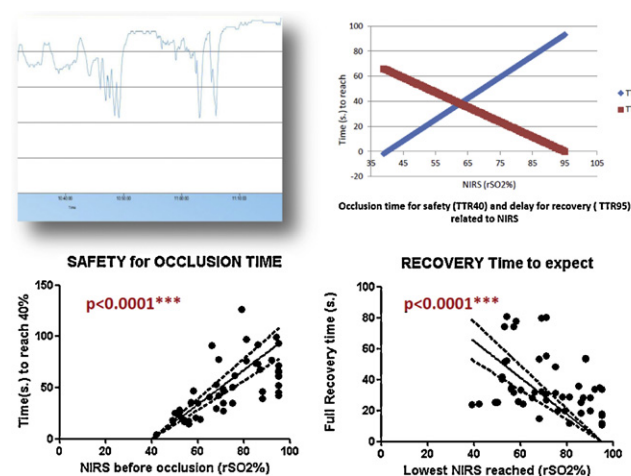
Methods We prospectively enrolled 20 consecutive children (mean \pm SD: age = 2.8 ± 3.4 y.o.; weight = 15.6 ± 13.6 kg) who underwent transcatheter balloon dilation of pulmonary valve, trunk or conduit stenosis. Procedure was done under general anesthesia with regular monitoring. SpO2 was used by physician for patient safety. Cerebral oxygen saturation (Rso2) was monitored by NIRS. Each brain desaturation events were collected. 40% of rSO2 was considered as the cut-off limit below which brain injury may occurs. Time to reach this limit (TTR40), time to recover (TTR95), area under curve and deepness of the events were extracted (Appendix 1).

Results The total number of inflation was 56, with a mean of 2.8 inflations per patient (min = 2; max = 10). The NIRS curve had two parts: it decreased during balloon inflation due to CO interruption and then increased during the reperfusion. Both were easily detected (Se = 100%) whereas the SpO2 remained wrongfully high. Near 36% of desaturation were under the cut-off (NIRS < 40%). TTR40 was 56.76 s and TTR95 was 34.85 s for the whole group. The linear regression based on NIRS shows two significant ($P < 0.0001$) slopes: -0.68 rSO2%/s for TTR40 and $+0.70$ rSO2%/s for TTR95. Our results suggest that a cumulative time over 44 s (95% CI) of multiples occlusions without recovery may cause brain injury. A recovery delay of 40 s (95% CI) after each set of balloon inflation is therefore mandatory.

Conclusion Pulmonary valve, trunk or conduit dilatation requires short occlusion and total recovery before any new inflation.

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Appendix 1



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